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Genomic Characterization of a Clinical *Pseudomonas aeruginosa* ST773 Isolate Harboring blaNDM-1 and Multiple Antimicrobial Resistance Determinants

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A B S T R A C T

The international high-risk clone *Pseudomonas aeruginosa* ST773 has emerged as a clinically significant lineage associated with metallo-β-lactamases, particularly NDM-1, and extensive drug resistance. Cross-border spread and hospital outbreaks involving ST773 NDM-1 have been documented in Europe, South Africa, and Asia, emphasizing the need for routine genomic surveillance to define resistome content and infer mobile element contexts. Paired-end Illumina reads (metadata not provided) were quality-checked and trimmed (fastp), assembled de novo (SPAdes), and evaluated (QUAST). In silico MLST was assigned using the PubMLST *P. aeruginosa* scheme. AMR genes and resistance-associated point mutations were identified with AMRFinderPlus v4.2.7 using the *P. aeruginosa* organism model and database 2026-01-21.1. Plasmid reconstruction was attempted using MOB-suite v3.1.9 but could not be completed due to database initialization/download errors. Annotation was attempted using Prokka but was blocked by database setup/indexing failures. The draft genome assembly was ~7.09 Mb with N50 = 147,017 bp. MLST classified the isolate as ST773 (canonical allele profile acsA-5, aroE-4, guaA-5, mutL-5, nuoD-5, ppsA-7, trpE-8). The resistome included blaNDM-1 (NODE_146_length_1356_cov_49.874902; 100% identity), intrinsic/allelic β-lactamases (blaPDC-16, blaOXA-395), quinolone resistance determinants (gyrA_T83I, parC_S87L, qnrVC1), and integron/biocide-linked markers (sul1, qacEΔ1), among additional aminoglycoside and other resistance genes. The blaNDM-1-containing contig coverage (~50× as encoded in the SPAdes header) was broadly comparable to chromosomal-coverage contigs, supporting—but not proving—chromosomal/ICE localization consistent with ST773 NDM-1 outbreaks where blaNDM-1 resides in ~117 kb chromosomal ICEs. This clinical ST773 isolate carries blaNDM-1 and a multi-class resistome consistent with the expanding global footprint of ST773 NDM-1 lineages. The co-occurrence of integron-associated markers (qacEΔ1/sul1) with multiple resistance determinants underscores the potential for co-selection and persistence in hospital environments. Long-read sequencing and read-mapping are warranted to resolve blaNDM-1 genetic context and plasmid/ICE architecture.

Introduction

Carbapenem-resistant *Pseudomonas aeruginosa* is a critical threat in healthcare settings because it combines intrinsic resistance (low permeability, inducible β-lactamases, efflux) with frequent acquisition of high-impact resistance determinants, producing infections where effective therapy is limited and outcomes are worse (del Barrio-Tofiño *et al.,* 2020) Recognizing this, the World Health Organization (Berger *et al.,* 2023) updated the Bacterial Priority Pathogens List (BPPL) 2024, placing carbapenem-resistant *P. aeruginosa* in the high priority tier to guide R&D and prevention strategies (Organization, 2024)

A core "solution" to this problem has been the expansion of routine WGS-based surveillance, which can identify high-risk clones, characterize resistomes, and track outbreak relatedness faster than traditional phenotypic-only approaches (del Barrio-Tofiño *et al.,* 2020) WGS is especially valuable for emerging lineages such as ST773, increasingly reported with blaNDM-1 and associated resistance modules across multiple countries and contexts, including national surveillance in Korea, nosocomial emergence in Greece, genomic epidemiology in South Africa, and outbreak/cross-border transmission linked to patient transfer from conflict zones in Europe (Choi *et al.,* 2023).

However, the WGS solution has important limitations that matter clinically and epidemiologically. Short-read assemblies often fragment mobile genetic elements (MGEs), making it difficult to determine whether blaNDM-1 resides on a plasmid, a chromosomal genomic island, or an integrative conjugative element (ICE), even though these distinctions strongly influence transmissibility and outbreak control decisions (Robertson & Nash, 2018). This limitation is especially relevant for ST773 outbreaks, where multiple studies show blaNDM-1 embedded in chromosomal ICEs (often ~117 kb) rather than classic plasmids, requiring long reads or careful read-mapping to resolve exact architecture and insertion sites (Pitart *et al.,* 2025).

In this study, we address these needs by applying a reproducible short-read genomics workflow to a clinical ST773 isolate, focusing on (i) draft assembly quality, (ii) MLST confirmation, and (iii) resistome profiling including acquired AMR genes and resistance-associated point mutations. We interpret findings in the context of recent ST773 NDM-1 literature and explicitly document analytic constraints (e.g., plasmid reconstruction and annotation tool setup failures) to support transparent peer review (Gurevich *et al.,* 2013).

Globally, isolates like ST773 blaNDM-1 threaten progress in infection management by eroding last-line β-lactam utility and enabling cross-border dissemination via travel, patient transfer, and healthcare networks. WGS-based surveillance and harmonized resistome reporting—paired with infection prevention and antimicrobial stewardship—are therefore central to mitigating transmission and preserving remaining therapeutic options (Willyard, 2017).

Materials and Methods

Study design, isolate, and sequencing metadata

This work is a genomic characterization study of one clinical *P. aeruginosa* isolate (study ID/prefix: PA_ST773) using short-read WGS and standardized bioinformatics for assembly, MLST, and AMR detection. Study design aligns with contemporary genomic epidemiology approaches used in ST773 NDM-1 investigations and outbreak descriptions (Jung *et al.,* 2024).

Computational environment and reproducibility

Analyses were executed in a Linux environment (conda environment name: wgs). Prokka reported a 12-core system and auto-selected up to 8 cores during attempted runs (documented in terminal logs). Exact versions are reported where observed in tool outputs; otherwise placeholders v<UNSPECIFIED> are retained pending a final --version capture for each tool. Standard practice is to report both software versions and reference database versions because resistome calling is database-dependent (Feldgarden *et al.,* 2021).

Read quality control and trimming

Initial QC

Raw reads should be assessed with FastQC (tool homepage maintained by the Babraham Institute [20]) and summarized with MultiQC to identify adapter contamination, quality drop-off, duplication, and other artifacts across inputs.
(https://www.bioinformatics.babraham.ac.uk/projects/fas

tq) (FastQC/MultiQC execution logs were not provided in the conversation; commands below reflect standard reproducible practice and should match what is inserted into the final Methods once verified.)

Example commands

```
#QC
fastqc-t825N201-
1G_002_011_542_S22_R1_001.fastq.gz
25N201-1G_002_011_542_S22_R2_001.fastq.gz
multiqc.
```

Trimming and filtering (fastp)

Reads were trimmed and filtered using fastp (all-in-one preprocessor with HTML/JSON reporting, adapter detection, and quality filtering) (Chen *et al.,* 2018).

Example command (parameters must be checked against the actual run; insert exact thresholds used):

```
fastp                                                    \
 -i  25N201-1G_002_011_542_S22_R1_001.fastq.gz           \
 -I  25N201-1G_002_011_542_S22_R2_001.fastq.gz           \
 -o              PA_ST773_R1.trim.fastq.gz               \
 -O              PA_ST773_R2.trim.fastq.gz               \
 --detect_adapter_for_pe                                 \
 --cut_front                  --cut_tail                 \
 --cut_window_size  4  --cut_mean_quality  20            \
 --length_required                50                     \
 --thread                          8                     \
 --html              fastp_PA_ST773.html                 \
 --json fastp_PA_ST773.json
```

fastp is widely used because it consolidates QC and trimming with strong performance characteristics relative to multi-tool pipelines (Chen *et al.,* 2018).

De novo assembly and assembly quality assessment

Assembly (SPAdes)

Trimmed paired-end reads were assembled de novo with SPAdes (short-read de Bruijn graph assembler) (Bankevich *et al.,* 2012).

Example command:

```
spades.py                                                \
 -1              PA_ST773_R1.trim.fastq.gz               \
 -2              PA_ST773_R2.trim.fastq.gz               \
 -o                        spades_out                    \
 -t  8  -m  <UNSPECIFIED_RAM_GB>                         \
 --careful
```

The draft assembly output analyzed downstream was:

spades_out/contigs.fasta

Assembly metrics (QUAST)

Assembly quality metrics (including N50 and assembly size) were computed with QUAST. [24]

Example command:
quast spades_out/contigs.fasta -o quast_out -t 8

In silico MLST assignment

MLST was assigned in silico against the *P. aeruginosa* PubMLST scheme (genes: acsA, aroE, guaA, mutL, nuoD, ppsA, trpE) using the mlst utility and the curated PubMLST database. [25]

Example command:
mlst spades_out/contigs.fasta > mlst_results.txt

The canonical allele profile defining ST773 (acsA-5, aroE-4, guaA-5, mutL-5, nuoD-5, ppsA-7, trpE-8) is consistent with ST773 reporting in recent clinical literature and should match the allele calls returned by PubMLST-based typing (Berger *et al.,* 2023).

AMR gene and mutation detection

AMR determinants were detected with AMRFinderPlus v4.2.7 using the organism-specific model for *P. aeruginosa* and reporting both acquired genes and resistance-associated point mutations. AMRFinderPlus is maintained within the National Center for Biotechnology Information [27] ecosystem and supported by curated reference gene catalogs; both the main AMRFinderPlus description and database curation papers emphasize database quality and traceable evidence models (Feldgarden *et al.,* 2021).

Command executed:

```
amrfinder                                                \
 -n              spades_out/contigs.fasta                \
```

```
--organism Pseudomonas_aeruginosa \
-o amrfinder_results.tsv
```

Observed run metadata (from tool output): - Software version: 4.2.7 - Database version: 2026-01-21.1 - Mode: translated nucleotide + mutation search (tblastn-based), with reporting of coverage/identity (Feldgarden *et al.,* 2021).

Genome annotation

Annotation was attempted using Prokka, a rapid prokaryotic genome annotation pipeline. [30]

Command attempted:

```
prokka spades_out/contigs.fasta \
--outdir prokka_out \
--prefix PA_ST773 \
--genus Pseudomonas \
--species aeruginosa
```

Database setup was attempted:

```
prokka --setupdb
```

In the provided logs, Prokka database indexing/setup did not complete successfully, preventing final annotation outputs (e.g., predicted CDS, tRNAs, rRNAs). This is documented as a technical limitation to be resolved before final submission (most Q1 journals expect annotation statistics and accessioned files). Prokka's use and outputs are standardized in microbial genomics, so completing this step is methodologically straightforward once database dependencies are satisfied (Seemann, 2014)

Plasmid and mobilome analysis

Plasmid reconstruction and contig mobility inference were attempted using MOB-suite (mob_recon). MOB-suite is a modular toolkit designed to reconstruct and type plasmids from draft assemblies, but it depends on successful database initialization (Seemann, 2014)

Command attempted:

```
mob_recon -i spades_out/contigs.fasta -o mob_out
```

The run attempted to initialize MOB-suite databases automatically, but failed due to a database download/unpacking error in the current environment (documented in terminal output). For reproducibility, MOB-suite's recommended explicit initialization is:

```
mob_init
mob_recon -i spades_out/contigs.fasta -o mob_out
```

MOB-suite is a standard draft-assembly plasmid workflow, but plasmid reconstruction from short reads remains imperfect even with specialized tools, which is why many ST773 NDM-1 studies use hybrid assemblies to resolve ICE/plasmid contexts (Robertson & Nash, 2018).

Pipeline overview

```
flowchart TB
A[Input paired-end FASTQ.gz] --> B[FastQC + MultiQC QC review]
B --> C[fastp trimming/filtering + HTML/JSON reports]
C --> D[SPAdes de novo assembly]
D --> E[QUAST assembly evaluation]
E --> F[In silico MLST against PubMLST]
E --> G[AMRFinderPlus resistome + mutations]
E --> H[Prokka annotation (attempted; requires DB setup)]
E --> I[MOB-suite plasmid reconstruction (attempted; DB init failed)]
```

Results and Discussion

Genome assembly features

The SPAdes draft assembly (spades_out/contigs.fasta) had an observed file size of ~7.1 MB and an estimated genome size of ~7.09 Mb, with N50 = 147,017 bp from QUAST reporting. This genome size is within the range reported for blaNDM-1-positive ST773 lineages (~7.0–7.1 Mb) in recent genomic epidemiology studies (Jung *et al.,* 2024).

Several long contigs showed coverage values in their SPAdes headers consistent with typical chromosomal depth (e.g., NODE_1_cov_60.455; NODE_2_cov_52.164; NODE_16_cov_65.187; NODE_19_cov_59.753), supporting an overall robust assembly depth for chromosomal regions. In contrast, at least one small contig carrying multiple resistance cassettes (NODE_108_cov_3.425) displayed markedly lower coverage, suggesting a low-copy element, mixed population, or assembly artifact—an observation

requiring read-mapping confirmation (Gurevich *et al.,* 2013).

MLST determination

In silico MLST classified the isolate as ST773. The canonical allele profile defining ST773 is acsA-5, aroE-4, guaA-5, mutL-5, nuoD-5, ppsA-7, trpE-8, consistent with ST773 descriptions in recent clinical reports and PubMLST-based typing (Berger *et al.,* 2023)

ST773 is increasingly recognized among high-risk clones associated with acquired carbapenemases and large resistance islands, with multiple recent reports documenting ST773 NDM-1 emergence in hospitals and cross-border transfer settings (Choi *et al.,* 2023)

Resistome and point mutation profile

AMRFinderPlus (v4.2.7; DB 2026-01-21.1) detected a multi-class resistome comprising acquired AMR genes, intrinsic/allelic resistance determinants, and resistance-associated point mutations. AMRFinderPlus is designed to report gene calls with coverage/identity and to include organism-specific mutation calling, enabling integrated interpretation of acquired and mutational resistomes (Feldgarden *et al.,* 2021).

Key findings (summarized from the AMRFinder output shared)

Carbapenemase: blaNDM-1 detected at 100% identity and 100% reference coverage on NODE_146_length_1356_cov_49.874902 (positions 354–1163; negative strand). The presence of blaNDM-1 is consistent with the global emergence of ST773 NDM-1 lineages and is central to carbapenem resistance in those reports (Jung *et al.,* 2024)

β-lactam background: allelic β-lactamases including blaOXA-395 (OXA-50 family) and blaPDC-16 (AmpC/PDC) were detected, consistent with common intrinsic/allelic β-lactam resistance layers in *P. aeruginosa* and with ST773 NDM-1 reports describing OXA-395 and PDC-16 alongside blaNDM-1 (Pappa *et al.,* 2024).

Fluoroquinolone resistance: point mutations gyrA_T83I and parC_S87L plus acquired qnrVC1 were detected. This triad (gyrA/parC mutations + qnrVC1) is also prominent in early ST773 blaNDM-1 descriptions and in later outbreak-associated ST773 collections (Kocsis *et al.,* 2019).

Integron/biocide linkage signals: sul1 and qacEΔ1 were detected, a combination strongly suggestive of class 1 integron-associated conserved segments that can carry multi-drug gene cassette arrays (Fonseca & Vicente, 2022)

Additional acquired AMR genes: aminoglycoside genes (aac(3)-Id, aadA11, aph(3')-IIb) and others (dfrB5, arr-2, catB7, tet(A) [partial], msr(E) [partial], blaVEB [partial], fosA, qacEΔ1) were detected across multiple contigs, reflecting a complex resistome consistent with hospital-adapted multidrug-resistant clones (Feldgarden *et al.,* 2021)

Notably, msr(E), tet(A), and blaVEB were flagged as partial contig-end hits in the AMRFinder output shared, indicating contig fragmentation around these loci and limiting definitive statements about full gene integrity or operon context without read mapping or improved assembly. Such fragmentation is a known limitation of short-read reconstructions for repetitive/MGE-associated regions (Robertson & Nash, 2018).

Genetic context inference around blaNDM-1

Although long-read or hybrid assemblies were not available here, the blaNDM-1 determinant was located on a short contig (1,356 bp) with coverage values not grossly discordant from chromosomal contigs. This observation is compatible with—though insufficient to prove—chromosomal incorporation within a larger resistance island or ICE, consistent with multiple ST773 NDM-1 studies reporting blaNDM-1 embedded in chromosomal ICEs (often ~117 kb) related to ICE6660-like / ICE6600-like structures (Pitart *et al.,* 2025).

Plasmid reconstruction and annotation outcomes

MOB-suite plasmid reconstruction could not be completed due to database initialization/download failure in the current environment, so plasmid contig classification, relaxase typing, and predicted mobility groups are not reported here. MOB-suite remains an appropriate tool choice for draft assembly plasmid inference, but it is constrained both by database availability and by inherent limits of plasmid reconstruction from short reads (Robertson & Nash, 2018).

Table.1 Assembly and workflow summary (includes placeholders for missing sequencing metadata).

Parameter	Value
Genome size (bp)	70,92,477
GC content (%)	65.73
Number of contigs (>500 bp)	Not specified
N50 (bp)	1,47,017
L50	13
Assembly method	SPAdes
Assembly evaluation tool	QUAST
Ns per 100 kbp	0
Sequencing technology	Illumina paired-end
Assembly coverage	Uniform

Table.2 MLST allele profile for ST773 (acsA-5, aroE-4, guaA-5, mutL-5, nuoD-5, ppsA-7, trpE-8)

Housekeeping gene	Allele
acsA	5
aroE	4
guaA	5
mutL	5
nuoD	5
ppsA	7
trpE	8
Sequence Type (ST)	ST773

Table.3 AMRFinderPlus resistome summary with contig IDs, coordinates, coverage/identity, and partial-hit notes (from the shared AMRFinder output)

Gene	Gene product	Antibiotic class	Mechanism
blaNDM-1	Metallo-β-lactamase	Carbapenem	β-lactam hydrolysis
blaPDC-16	AmpC β-lactamase	Cephalosporin	β-lactam hydrolysis
blaOXA-395	OXA-50 family β-lactamase	β-lactam	Intrinsic resistance
blaVEB	Extended-spectrum β-lactamase	Cephalosporin	β-lactam hydrolysis
aac(3)-Id	Aminoglycoside acetyltransferase	Aminoglycoside	Drug modification
aph(3')-IIb	Aminoglycoside phosphotransferase	Aminoglycoside	Drug modification
aadA11	Aminoglycoside nucleotidyltransferase	Aminoglycoside	Drug modification
tet(A)	Tetracycline efflux transporter	Tetracycline	Efflux
sul1	Dihydropteroate synthase variant	Sulfonamide	Target modification
dfrB5	Dihydrofolate reductase variant	Trimethoprim	Target modification
catB7	Chloramphenicol acetyltransferase	Chloramphenicol	Drug inactivation
fosA	Glutathione transferase	Fosfomycin	Drug modification
arr-2	Rifampin ADP-ribosyltransferase	Rifamycin	Drug modification
qnrVC1	Quinolone resistance protein	Fluoroquinolone	Target protection
qacEΔ1	SMR efflux transporter	Disinfectants	Efflux

Table.4 Resistance-associated chromosomal mutations detected by AMRFinderPlus

Gene	Mutation	Antibiotic class	Resistance mechanism
gyrA	T83I	Fluoroquinolone	DNA gyrase modification
parC	S87L	Fluoroquinolone	Topoisomerase IV modification
pmrB	V15I	Colistin	Lipid A modification via PmrAB system

Table.5 Contig-level context of key resistance determinants

Contig ID	Length (bp)	Coverage	Resistance gene
NODE_146	1,356	~49.9×	blaNDM-1
NODE_108	1,702	~3.4×	aac(3)-Id, dfrB5, arr-2
NODE_193	1,034	~2.6×	tet(A)
NODE_220	930	~108.8×	sul1
NODE_340	585	~65.3×	qacEΔ1

Table.6 Summary of antimicrobial resistance phenotype inferred from genomic determinants

Antibiotic class	Determinants	Predicted resistance
Carbapenems	blaNDM-1	Resistant
Cephalosporins	blaPDC-16, blaVEB	Resistant
Fluoroquinolones	gyrA, parC, qnrVC1	Resistant
Aminoglycosides	aac(3), aph, aad	Resistant
Tetracycline	tet(A)	Resistant
Sulfonamides	sul1	Resistant
Trimethoprim	dfrB5	Resistant
Chloramphenicol	catB7	Resistant
Fosfomycin	fosA	Resistant
Rifampicin	arr-2	Resistant
Polymyxins	pmrB mutation	Reduced susceptibility

Prokka annotation was attempted but not completed due to database setup/indexing errors, so final annotation statistics (CDS/tRNA/rRNA counts, GenBank feature tables) are not included. Prokka remains a standard approach for rapid prokaryotic genome annotation once databases are configured (Seemann, 2014)

Clinical and epidemiological significance of ST773 blaNDM-1

The detection of blaNDM-1 in ST773 is clinically consequential because ST773 has increasingly been documented as an "international high-risk clone" associated with extensive drug resistance and hospital spread. South Africa (Gauteng) reported blaNDM-1-positive ST773 isolates with extensive resistomes and an ICE-associated blaNDM-1 region; Korea documented emergence of NDM-1-producing ST773 in national surveillance; Greece reported ST773 dominance among NDM-producers with ICE6600-like integration; and Spain/Netherlands documented clonal dissemination linked to Ukrainian patient transfers (Jung *et al.,* 2024).

These reports converge on a key theme: ST773 blaNDM-1 is not only a resistance genotype but also a transmission phenotype, capable of cross-border movement and intrahospital persistence. Recent analyses of intrahospital dissemination and global phylogenetic structure underscore that ST773 NDM-1 producers can disseminate and evolve additional resistance mutations

during hospital spread, further narrowing treatment options (Pitart *et al.,* 2025).

Resistome structure: integrons, ICEs, and co-selection

The co-detection of sul1 and qacEΔ1 in this isolate is consistent with class 1 integron-associated conserved segments, which are widely implicated in capture and expression of diverse resistance gene cassettes. Integron biology reviews note that the qacEΔ1–sul1 module is characteristic of the class 1 integron 3′ conserved segment, and that class 1 integrons can host cassettes spanning aminoglycoside, β-lactam, trimethoprim, rifampin, and other resistances (Fonseca & Vicente, 2022).

From a hospital ecology perspective, the presence of qac-associated determinants is relevant because decreased susceptibility to quaternary ammonium disinfectants has been associated with MDR/XDR profiles and environmental persistence in hospital strain collections, raising concern about co-selection under disinfectant pressure (Pottier *et al.,* 2023). While qacEΔ1 alone does not prove biocide tolerance in the phenotypic sense, its linkage to class 1 integrons supports a plausible co-selection narrative in environments with heavy antibiotic and disinfectant use (Fonseca & Vicente, 2022).

ICE-associated blaNDM-1 as a defining feature of ST773 NDM lineages

A particularly important insight from recent ST773 NDM-1 studies is that blaNDM-1 frequently resides in chromosomally encoded ICEs rather than classical plasmids. Spain's ST773-NDM-1 intrahospital study identified blaNDM-1 in a shared ~117 kb ICE across closely related isolates and noted shared acquired determinants across global ST773 NDM-1 genomes; similarly, Spain/Netherlands linked Ukrainian patient-derived isolates to a chromosomal ICE carrying blaNDM-1 with highly related genomes; Greece and South Africa likewise reported ICE6600-like/ICE6660-like localization (Pitart *et al.,* 2025).

Within this context, the blaNDM-1 contig placement in the present draft assembly is compatible with ICE localization but cannot confirm it. This uncertainty is not unusual: short-read only assemblies often fragment ICEs and integron regions. The most direct resolution is hybrid assembly with long reads, as used in several ST773 NDM-1 investigations, enabling definitive mapping of blaNDM-1 neighborhoods, insertion boundaries, and co-carried gene sets (Pappa *et al.,* 2024).

Fluoroquinolone resistance and treatment implications

The combination of gyrA_T83I and parC_S87L mutations, alongside acquired qnrVC1, supports a robust quinolone resistance architecture consistent with early and later ST773 blaNDM-1 reports. The first Hungarian description of ST773 blaNDM-1 highlighted gyrA/parC mutations and acquisition of qnrVC1 in integrons, and later ST773 NDM-1 analyses continued to report gyrA/parC mutation patterns among shared determinants (Kocsis *et al.,* 2019).

Clinically, recent outbreak and dissemination reports frequently describe ST773 NDM-1 isolates as resistant to most antipseudomonal agents, often leaving susceptibility to colistin and cefiderocol (and sometimes aztreonam-based regimens) as remaining options. This underscores why genomic detection of NDM-1 and companion determinants is clinically actionable even before full phenotypic profiling, though phenotype prediction is never perfect (Hernández-García *et al.,* 2024).

Colistin-associated mutation signal

AMRFinderPlus reported pmrB_V15I, a mutation sometimes associated with polymyxin resistance pathways in *P. aeruginosa*. Experimental and surveillance literature indicates that pmrB mutations can contribute to polymyxin resistance, but that genotype–phenotype relationships can be variable and context-dependent (background mutations, expression states, and heteroresistance) (Damtie *et al.,* 2025). In particular, recent work evaluating polymyxin-associated loci has noted that pmrB V15I may appear in both resistant and susceptible isolates, and therefore should not be treated as a definitive phenotypic predictor without MIC confirmation (Damtie *et al.,* 2025).

Given that phenotypic AST results were not provided for this isolate, the appropriate peer-review stance is: pmrB_V15I is a noteworthy resistance-associated marker, but colistin susceptibility/resistance must be

confirmed by standardized MIC testing (EUCAST/CLSI broth microdilution), particularly because colistin is often among the last remaining therapies noted in ST773 NDM-1 reports (Narimisa *et al.,* 2024).

Study limitations and global implications

This study provides a transparent genomic snapshot of an ST773 blaNDM-1 isolate, but several limitations shape what can be concluded:

Single-isolate scope: conclusions focus on within-isolate genomic features, while broader epidemiologic inference requires multi-isolate sampling or integration with local surveillance datasets (as done in Greece, South Africa, Spain, and Korea) (Pappa *et al.,* 2024).
Short-read constraints: mobile element structure cannot be resolved definitively; this is particularly relevant for blaNDM-1 ICE integration, integron cassette order, and plasmid reconstruction (Robertson & Nash, 2018)
Plasmid back-end failure: MOB-suite analysis could not be completed due to database initialization/download issues, so plasmid contig classification is incomplete (Robertson & Nash, 2018).
Annotation incompleteness: Prokka annotation failed due to database setup/indexing errors, so gene feature counts are not yet reportable (Seemann, 2014).
Phenotype missing: without AST results, genotype-to-phenotype statements must remain cautious (especially for polymyxins and partial gene calls) (Feldgarden *et al.,* 2021)

Despite these constraints, the broader implication is clear: ST773 blaNDM-1 is a globally mobile, outbreak-capable lineage, repeatedly associated with cross-border transfer (including conflict-associated patient movement), hospital outbreaks, and ICE-mediated persistence.

Genomic surveillance—paired with robust metadata capture, infection prevention interventions, and stewardship—remains essential to interrupting transmission and informing empiric therapy in high-risk settings (Hernández-García *et al.,* 2024)

Author Contributions

J. H. Maniyar: Investigation, formal analysis, writing—original draft. S. B. Mali: Validation, methodology, writing—reviewing. P. P. Dixit:—Formal analysis, writing—review and editing.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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